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SEM Study of Resorption Patterns in Dental Hard Tissues

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Introduction

Dental hard tissue resorption is an essential physiological process within the change from deciduous to permanent dentition. It further occurs in pathological processes found in periodontitis apicalis and marginalis, after apicotomia and within internal granuloma.

Objectives

As there is no literature concerning the micromorphological comparison on the different resorption patterns resulting from the different sources mentioned it was the aim of our study to show 1. whether there are differences between the resorption patterns resulting from different reasons and 2. if there are differences , how they may be described on the basis of this SEM study.

Material and Methods

Teeth extracted for the reasons mentioned above were dried in acetone, sputtered with gold palladium and investigated with SEM (Philips XL30 FEG) at 20 kV beam current. The tooth exhibiting the internal granuloma was prepared for light microscopy by decalcification, sectioning after embedding in paraffine and staining with Azan. The different objects were studied by SEM and the surface structure was described as to roughness, regularity/irregularity, structure and texture. They were compared taking into consideration the origin of the hard tissue resorption. Additionally on the basis of our light microscopic study of the internal granuloma cell types, the composition and structure of the resorbing tissue was defined.

Results

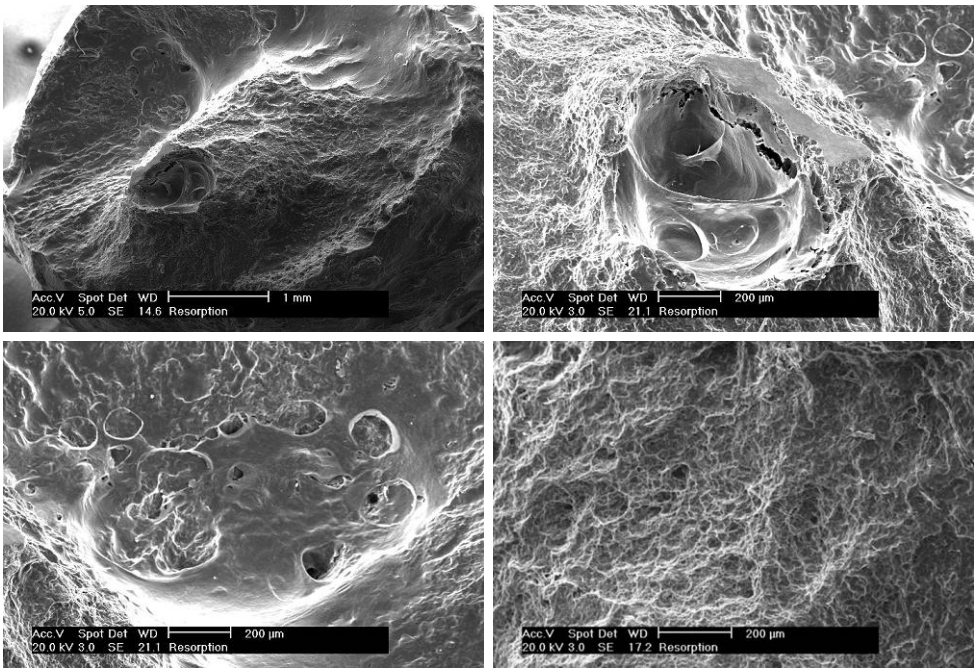

Fig 1a-d: Parodontitis apicalis acuta has led to an irregular (a) partly vast (c) partly polycyclic (d) dissolution of the dentin around the apex (b) leaving it unresorbed because of non-vital tissue within the root canal.

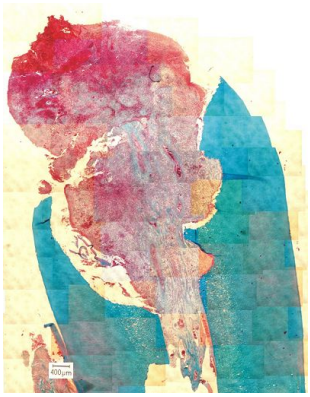

Fig. 2a: The histological overview of the internal granuloma reveals different tissue zones consisting of necrotic tissue, granulomatous tissue, highly vascularized fibrous tissue, resorptive tissue with fluent transition into normal vital root pulp tissue.

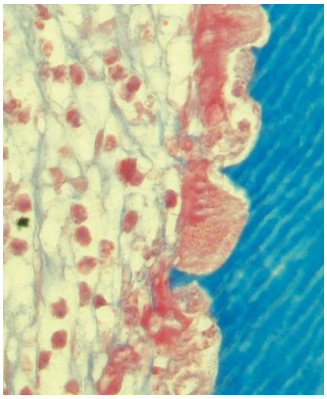

Fig. 2b: Higher magnification (225x) reveals the multinucleated odontoclasts within resorption lacunae surounded by vital pulpal fibroblasts.

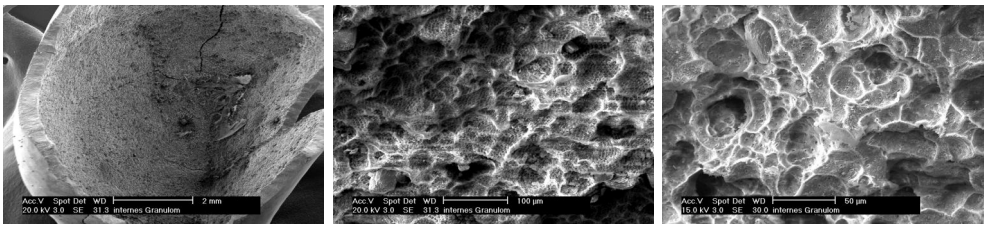

Fig. 3a-c: The coronal dental hard tissue has almost completely been resorbed by the internal granuloma just leaving an eggshell of enamel (a), within the deep lacunae showing the typical keyhole structure in fields of enamel resorption (b) and exposing open dental tubules in the dentin representing the aggressiveness of this pathological resorptive process.

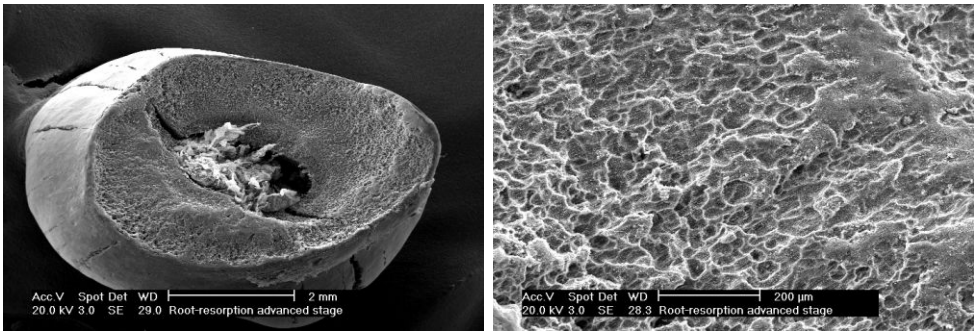

Fig. 4a+b.: The pattern due to physiological root resorption of a deciduous upper front tooth (a) shows very regular polycyclic shallow lacunae representing a uniform longterm ongoing process (b).

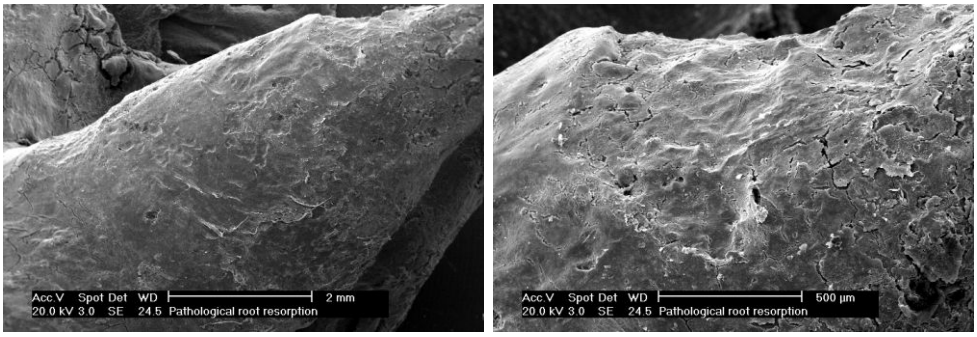

Fig. 5a+b: The shallow resorptions due to chronic periodontitis are covered by newly formed cementum and can be seen on the intrabony and extrabony rootsurface. Thus open dentin tubules cannot be seen.

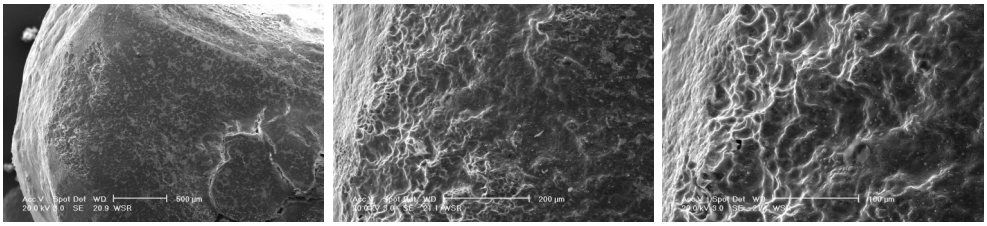

Fig. 6a-c: After root resection the apical resected surface (a) shows irregular polycyclic lacunae of different size (b) not exposing dental tubules (c).

Discussion

Internal root resorption is a rare remodelling process (Lyroudia et al. 2002) that in literature is mostly dealt with in case reports (Anil et al. 1993). On the basis of a light microscopic study of the whole tooth containing the internal granuloma, it was our intension to look at resorption patterns resulting from different pathological infammatory and physiological processes applying SEM.

Concordant with Bimstein et al. (1998) who could show resorption lacunae on the roots of primary deciduous teeth from children with prepubertal aggressive periodontitis we found intrabony and suprabony fields of root resorption. Odontoblasts and cementoblasts differ from osteoblasts in so far as they do not respond to hormones and cytokines that stimulate bone resorption. Hammarström et Lindskog (1992) therefore derive that the cementoblast or odontoblast layer has to undergo destruction due to nekrosis or inflammation before dental hard tissue resorption is about to occure. Thus it is concluded that there have to be further substances probably expressed by the reduced ameloblastic epithelium that make resorption of deciduous teeth possible. On the other hand it becomes obvious why inflammation in the case of periodontitis apicalis and marginalis or trauma as an etiological factor of the internal granuloma result in the resorption of dental hard tissues. Concerning ultrastructural features of odontoclasts resorbing enamel Sahara et al. (1998) could demonstrate that these tartrate-resistant acid phosphatase-positive multinucleated cells were quite similar to the cells resorbing dentin or cementum. Rich in mitochondria, lysosomes and free polysomes these cells secrete acids, organic components and hydrolytic enzymes phagocytosing enamel crystals then to be found in large cytoplasmic vacuoles which are later dissolved intracellularly. From a cell-biological standpoint this should be the same ongoing process in enamel resorption performed by the resorptive tissue of the internal granuloma.

Cytochemical studies revealed that H+ and K+ ATP-ase of odontoclast origin facilitate extracellular demineralization of the inorganic crystals, and hydrolytic enzymes such as trimetaphosphatase and p-nitrophenyl phosphatase are responsible for the intra- and extracellular digestion of the organic components of dentin and enamel such as collagenous fibres (Matsuda 1992).

Several studies have concentrated on resorption mechanisms due to molecular biological aspects. Kikuchi et al. (1989)saw 6.9 fold enhanced DNA synthesis in mesenchymal cells and 3.4 fold enhanced in epithelial cells gained from tissue resorbing deciduous teeth due to the application of Epidermal Growth Factor (EGF). Applying a dental hard tissue resorptive model Dreyer et Pierce (2001) could show that the lysosomal membrane antibody ED1 is a positive periodontal ligament cell (PDL) marker for mono- and multinucleated cells being involved in hard tissue resorption. Lossdorfer et al. (2002) assume that receptor activator of nuclear factor kappa B /and ligand (RANK/RANKL) which are cytokine-like proteins of the TNF-family play a central role in both physiological and pathological resorption. While odontoblasts, pulp fibroblasts PDL-fibroblasts and odontoclasts were RANKL positive, RANK positive immunostaining was detectable as well in multinucleated odontoclasts as in mononucleated precursors.

Our mikromorphological study comparing resorption patterns of different etiologies could show that though dental hard tissue resorption has been assumed to be ascribed to different cell types such as odontoclasts, cementoclasts or adamantoclasts, in the literature neigther on a morphological, ultrastructural, biochemical nor molecular biological basis can be distinguished into different cell types due to different biological sources of resorption, still these sources cause different morphological patterns of dental hard tissue resorption.

Conclusion

From our results we conclude that different sources causing resorption under physiological and pathological conditions lead to different morphologies in hard tissue resorption. In contrast to physiological resorption chronic inflammation of the periodont leads to irregular distict shallow resorption in areas with cement distruction, whereas acute inflammation causes agressive rapid deep resorption lacunae. This might also be of clinical and forensic interest.

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Abbreviations

ATP-ase: Adenosin Triphosphatase
EGF: Epidermal Growth Factor
PDL: Periodontal Ligament Cell
RANK/RANKL: Receptor Activation of Nuclear Factor B/ and Ligand
SEM: Scanning Elektron Microscopy
TNF: Tumor Nekrosis Factor

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